6.0 BASELINE HUMAN HEALTH RISK EVALUATION

Section 6.0 presents the methodology for and the results of a baseline human health risk assessment conducted for the Area I study areas described in Sections 1 through 5. The objective of the assessment is to estimate potential current or future risks to the public from the organic and inorganic chemicals detected in the surface water, soil, wetland soil, and sediment samples collected in the study areas. Section 6.1 provides an overview of the risk assessment process. Sections 6.2 through 6.5 outline the methodology used to conduct the baseline human health risk assessment. An analysis of the uncertainties common to the evaluation of all (or most) of the study areas is presented in Section 6.6. The results of the baseline risk assessment of area-specific chemical concentrations in the environmental media are presented in Sections 6.7 through 6.9. Section 6.10 presents a summary of the baseline human health risk evaluation. The risk assessment conducted for this report follows the most recent guidance from the EPA (EPA, 1989d and 1991a), including regional EPA guidance (EPA, 1989b, 1994f, 1995d, and 1996f).

6.1 Introduction - Overview of Risk Assessment Process

A risk assessment provides the framework for developing risk information necessary to assist in determining the need for remediation at a site and developing potential remedial alternatives for a site. A baseline human health risk assessment consists of five major components, as follows:

- Data evaluation and identification of chemicals of potential concern (COPCs),
- Exposure assessment,
- Toxicity assessment,
- Risk characterization, and
- Characterization of uncertainty in the risk estimates.

To assess potential public health risks, four major aspects of chemical contamination and exposure must be considered; contaminants with toxic characteristics must be found in

the environmental media; the contaminants must be released by either natural processes or by human action; potential exposure points must exist; and human receptors must be present at the point of exposure. Risk is a function of both toxicity and exposure. If any one of the requirements listed above is absent for a specific site, the exposure route is regarded as incomplete and no potential risks will be considered for human receptors.

The risk assessment for the Area I study area estimates the potential for human health risk at each of three study areas shown in Figure 1-2;

- Area A-1 - Morgan Francis Property
- Area A-2 - Commercial Properties west of Ferry Creek
- Area A-3 - Ferry Creek and properties east of Ferry Creek

The Data Evaluation Section is primarily concerned with the selection of COPCs that are representative of the type and magnitude of potential human health effects. Both current and historical data are considered in developing a list of COPCs for each medium. In turn, these COPCs are used to evaluate potential risks. A generic discussion of the process is contained in Section 6.2.1, and site-specific discussions are presented in Sections 6.7 through 6.9.

The Toxicity Assessment presents the available human health criteria for all the selected COPCs. This assessment is contained in Section 6.3; however, the final lists of study area COPCs are presented later in the document. This section is presented early to avoid repetition of the toxicity information because many COPCs are common to more than one of the study areas. Quantitative toxicity indices are presented where they are available. Enforceable standards such as Maximum Contaminant Levels (MCLs), regulatory guidelines such as Ambient Water Quality Criteria (AWQC) and Health Advisories, and dose-response parameters such as Reference Doses (RfDs) and Cancer Slope Factors (CSFs) are presented for each COPC.

The Exposure Assessment section (Section 6.4) identifies potential human exposure pathways at the study areas under consideration. Exposure routes are identified based on information on study area chemical concentrations, chemical release mechanisms, patterns of human activity, and other pertinent information to develop conceptual site models for each type of source. One overall set of exposure routes was developed for this report, but not all routes are applicable at all sites. Section 6.4.6 presents the equations and relevant input parameters for estimating chemical intake. The study area-specific risk assessments present only those routes relevant to each study area, and refer to Section 6.4.6 for the details on the estimation method.

The Risk Characterization section (Section 6.5) describes how the estimated intakes are combined with the toxicity information to estimate risks. The actual numerical results of this exercise are presented in the study area-specific sections of this report. General uncertainties associated with the risk assessment process are discussed qualitatively in Section 6.6. Uncertainties associated with a particular site are provided in the site-specific sections.

6.2 Data Evaluation Methodology

Data evaluation is a study area-specific task that uses a variety of information to determine which of the detected chemicals at a study area are most likely to present a risk to potential receptors. The end result of this qualitative selection process is a list of COPCs and representative exposure point concentrations for each medium. The rationale for the selection and/or exclusion of each detected chemical is presented in the site-specific sections, Sections 6.7 through 6.9. The methodology used to identify COPCs for the Area I RI Report is provided in Section 6.2.1. The methodologies used to determine exposure point concentrations for the selected COPCs are presented in Section 6.2.2.

6.2.1 Selection of Chemicals of Potential Concern

COPCs for the baseline human health risk assessments are limited to those chemicals that exceed a selection criterion. For this risk assessment, federal and state risk-based and health-based criteria were used to reduce the number of chemicals and exposure routes considered in a risk assessment. The premise of this screening step is that risk is typically dominated by a few chemicals and that, although dozens may actually be detected, many chemicals may contribute minimally to the total risk. The purpose of using federal and state criteria is to satisfy the potential concerns of each regulatory agency since similar federal and state criteria may not be developed using the same methodologies and exposure assumptions.

Maximum detected concentrations (in a single sample) at each study area and in each medium were compared to the risk-based and health-based screening criteria. If the maximum concentration exceeded any of the screening criteria, that chemical was retained as a COPC for all exposure routes involving that medium. For example, if barium was retained for soil, this chemical was evaluated as a COPC for both ingestion and dermal exposure routes.

In general, all available validated data and unvalidated field screening data for copper and lead from historical investigations and the recent (1997) sampling effort were used to identify COPCs for a study area. As provided in Appendix F-1, an evaluation of the field screening data for copper and lead indicate a good correlation between field-screening and fixed-base laboratory results for copper and lead. Consequently, the field screening data for lead and copper were used in the baseline risk assessment. Study area- and medium-specific COPC summary screening tables are provided in Sections 6.7 through 6.9. Field screening data (other than lead and copper), unvalidated data, and analytical results qualified as rejected, R, during the data validation process were not considered due to their potential unreliability. For soil, data obtained from excavated locations, soil collected from depths greater than 15 feet (the maximum assumed depth for potential human exposure

during excavation/construction based on the State of Connecticut definition of accessible soils), and composite soil samples were not used in the COPC selection process.

Essentially, two types of COPCs are identified in the baseline human health risk assessment: direct exposure COPCs and additional COPCs based on potential contaminant migration tendencies, i.e. groundwater protection benchmarks. Direct exposure COPCs are those chemicals detected at maximum concentrations in excess of criteria developed for the protection of direct human contact with a medium, e.g., risk-based EPA Region III COPC screening levels for soil and tap water ingestion. Residential soil and tap water risk-based concentrations were included in the screening criteria for selection of COPCs. This approach is quite conservative for a site where no residential use is anticipated and surface water is not potable due to salinity. The approach was taken to assure protection of nearby residents and to allow for the inclusion of chemicals that may produce marginal risks to be included in the quantitative assessment. Other health-based criteria, e.g., Connecticut Remediation Standard Regulations for pollutant mobility, are used to identify groundwater protection benchmarks based on likely contaminant migration pathways at Area I study areas. Only chemicals selected as COPCs based on comparisons to direct contact criteria were evaluated quantitatively in the baseline risk assessment. All criteria used to identify COPCs for solid environmental matrices (soil, wetland soils, and sediments) and aqueous environmental matrices (surface water) are presented in Table 6-1 and Table 6-2, respectively. As discussed previously, the groundwater resource at Ferry Creek is not evaluated in this baseline risk assessment.

A discussion of the criteria used for COPC selection is provided in the remainder of this section, on a medium-specific basis.

6.2.1.1 Soils/Wetland Soils/Sediments

The solid matrix samples from the Area I study areas have been divided into soils, wetland soils, and sediments for the purposes of human health exposure evaluation. This breakdown is useful in distinguishing samples to which specific receptor groups may be

exposed. Soils are defined as solid matrix samples collected from relatively dry areas located outside designated wetland boundaries and not associated with creeks, creek beds, or the Housatonic River. Wetland soils are defined as solid matrix samples collected from within designated wetland boundaries. It should be noted that these samples may have been designated as either soils or sediments in earlier reports or by other contractors. Sediments are defined as solid matrix samples collected from creeks, creek beds, or the Housatonic River.

COPCs for solid environmental matrices (soils, wetland soils, and sediments) were selected for each of the Area I study areas. The COPCs selected for shallow soils, wetland soils, and sediments from depths of 0 to 2 feet bgs are presented separate from COPCs selected for "all soils". The "all soil" category refers to soil and sediment samples collected from depths of 0 to 15 feet bgs and is used to account for soil to which commercial workers may be potentially exposed. Soils to a depth of 15 feet are considered "accessible" by the State of Connecticut. If a chemical is identified as a COPC for shallow soils and sediments, it is automatically retained as a COPC for "all soil." If a compound is found in the subsurface soil only at a concentration of concern (in excess of a screening criteria), it is retained as a COPC for the "all soil" category only. All sample locations within a given area were used to determine COPCs for the area without regard to later division of the area data into subsets of data based on receptor locations.

The following screening criteria were used to identify COPCs for direct contact exposure to soils, wetland soils, and sediments;

- **EPA Region III COPC Screening Levels for Residential Soil Ingestion.** Although current and likely future land use within the Area I study areas is commercial/industrial and recreational, risk-based concentrations for soil ingestion for residential land use were used as a conservative approach. These values were developed using the current EPA Region III Risk-Based Concentration (RBC) Table (EPA, 1998b), which identifies concentrations of potential concern for nearly 600 chemicals in various media (air, drinking water, fish tissue, and soil) using certain

reasonably maximum exposure default assumptions. The EPA Region III residential soil ingestion values were calculated by assuming that a receptor is exposed to soil for 350 days per year for a 30 year exposure period. For carcinogenic chemicals, the values used for COPC screening are based on a 1E-6 target incremental lifetime cancer risk and incorporate age-adjusted factors (for small children and adults). The criteria for noncarcinogenic chemicals are based on a target hazard quotient (HQ) of 1.0. These EPA Region III residential soil ingestion values for noncarcinogenic chemicals were adjusted to COPC screening levels based on a target hazard quotient (HQ) of 0.1, which is one-tenth of the suggested cumulative target noncarcinogenic risk for a potential receptor, and exposure defaults for small children. The estimation of cumulative target noncarcinogenic risks is described in greater detail in Section 6.5.

- **EPA Soil Lead Guidance.** EPA Region III has not developed risk-based concentrations for lead. Although current and likely future land use within the Area I study areas is commercial/industrial and recreational, risk-based lead concentrations for soil ingestion for residential land use were used as a conservative approach. OSWER soil screening level of 400 mg/kg for residential land use (EPA, 1994) and the state screening levels of 500 mg/kg for residential exposure and 1,000 mg/kg for industrial exposure were used for COPC screening. The EPA's Integrated Exposure Uptake and Biokinetic (IEUBK) model which estimates the risk to a child resident is the basis for the OSWER residential soil screening level.

- **National Emission Standards for Hazardous Air Pollutants Benchmark for Asbestos.** EPA Region III has not developed risk-based concentrations for asbestos. Asbestos was a primary component of friction materials, e.g., gaskets material, sheet packing and friction materials, including clutch facing, transmission plates, and brake linings, manufactured at the Raymark facility. Quantitative risk estimates (inhalation risk estimates) cannot be developed for this parameter, however asbestos is considered a potential inhalation hazard. The National Emission Standards for Hazardous Air

Pollutants - EPA Regulation 40 CFR Subpart M, Part 61 (NESHAP) defines asbestos as material containing more than one percent asbestos. Since asbestos was detected at the site, TtNUS has adopted the NESHAP benchmark of one percent for an asbestos screening value.

- **Connecticut Remediation Standard Regulations (RSRs) for Direct Exposure (Residential and Industrial).** Connecticut RSRs for direct exposure to soil under residential and industrial land use are presented in the COPC screening tables. Although the standards for residential direct exposure are the limiting factor for COPC selection (values for residential exposure are less than those for industrial exposure), both of these standards are provided for informational purposes. RSRs for direct exposure are calculated using methodologies similar to those used to develop the EPA Region III COPC Screening Levels for soil ingestion. However, reasonable maximum exposure default assumptions employed by the state are slightly different than those advocated by EPA Region III (a residential receptor is assumed to be exposed to soil at a frequency of 365 days per year, instead of the EPA's 350 days per year assumption). The standards for carcinogenic chemicals are based on a 1E-6 target incremental lifetime cancer risk. The standards for noncarcinogenic chemicals are based on a target HQ of one. The State of Connecticut has not developed RSRs for all chemicals positively detected within the Area I study areas. For those chemicals lacking adopted RSRs, TtNUS has calculated RSRs (TtNUS, 1997e) using the methodologies outlined in the RSR guidance (CT DEP, 1996). These values were submitted to the state for review during the preparation of a report for the Lower Subbase Remedial Investigation for Naval Submarine Base, New London, and they were revised based on comments received from the state (B&RE, 1998). A summary of the RSRs developed by B&RE and used in this risk assessment are presented in Appendix F-2.

- **EPA Generic Soil Screening Levels (SSLs) for Transfers from Soil to Air (Inhalation).** EPA Generic SSLs (EPA, 1996d) for direct inhalation are used to evaluate chemicals that may volatilize from soil, as well as contaminated particulates that may be

present in air (fugitive dust) as a result of particulate entrainment from soil. The inhalation SSLs are calculated using default, residential land use exposure factors, infinite source models, and conservative default assumptions for source delineation. Therefore, these values are conservative and are designed to be protective of potential exposure at most sites. The EPA has calculated generic SSLs for approximately 110 organic and inorganic chemicals. SSLs for carcinogenic chemicals are based on a 1E-6 target incremental lifetime cancer risk. For noncarcinogenic chemicals, the SSLs are based on a target HQ of one.

Background concentrations for chemicals in soil, wetland soil, and sediment are presented in Appendix F-3. Metals concentrations in the background soils samples were used to select COPCs. Specifically, a noncarcinogenic metal detected in soils/wetland soils/sediments at a concentration greater than the COPC screening levels for soil, but equal to or less than the EPA Region III residential RBC for soil ingestion, AND the maximum detected background concentration was not selected as a COPC. A discussion of site data in comparison to the established inorganic and organic background levels is provided in each site-specific uncertainty section. It should be noted that background concentrations were considered when developing recommendations and conclusions for each site (identifying whether additional sampling or remediation is warranted).

Frequency of detection was used as a COPC selection criteria for parameters not known to be predominant study area contaminants (the predominant study area contaminants are PCBs, PAHs, dioxins, asbestos, and metals [especially copper, lead, and barium]). In general, other chemicals detected once or twice within a study area at a maximum concentration marginally exceeding a screening criteria were not selected as COPCs. The decisions to delete parameters on the basis of frequency of detection were reviewed with EPA Region I prior to the finalization of the COPC selection tables.

In order to identify potential contaminant migration to groundwater tendencies, the following criteria were used to evaluate shallow soil and "all soil" (soil collected from

depths of 0 to 15 feet bgs), but these criteria were not used to select COPCs for quantitative risk assessment:

- **EPA Generic Soil Screening Levels (SSLs) for Migration to Groundwater.** EPA Generic SSLs for migration to groundwater associated with a dilution and attenuation factor of 20 were also used to identify chemicals detected in soils/wetland soils/sediments at concentrations that may impact groundwater quality. The migration to groundwater SSLs are calculated using default, residential land use exposure factors, infinite source models, and conservative default assumptions for source delineation. Therefore, these values are conservative and are designed to be protective of potential exposure at most sites. The EPA has calculated generic SSLs for approximately 110 organic and inorganic chemicals. SSLs for carcinogenic chemicals are based on a 1E-6 target incremental lifetime cancer risk. For noncarcinogenic chemicals, the SSLs are based on a target HQ of one.

- **Connecticut RSRs for Pollutant Mobility (GB Classified Area).** The state has developed pollutant mobility RSRs for GA/GAA (drinking water source) and GB (non-drinking water source) classified areas. Since the Area I study area is classified by the state as a GB area, Connecticut RSRs for GB pollutant mobility were used to identify groundwater protection benchmarks. For most organic chemicals, RSRs for pollutant mobility are calculated using methodologies similar to those used to develop the EPA generic SSLs for migration to groundwater. However, the actual models and reasonable maximum exposure default assumptions employed by the state are different from those advocated by EPA Region III. The standards for carcinogenic chemicals are based on a 1E-6 target incremental lifetime cancer risk. The standards for noncarcinogenic chemicals are based on a target HQ of one. It should be noted that RSRs for inorganics, pesticides, and PCBs apply to SPLP or TCLP analytical results only. RSRs for these chemicals were used to identify groundwater protection benchmarks in the baseline human health risk assessment. The comparison of site data to these standards is presented in tables following the

COPC screening tables. As mentioned previously, the State of Connecticut has not developed RSRs for all chemicals positively detected at the Area I study areas. Therefore, B&RE has calculated RSRs using state guidance (CT DEP, 1996) for use in the risk assessment (Appendix F-2).

6.2.1.2 Surface Water

COPCs for surface water were selected using data for unfiltered samples. COPCs for direct exposure to surface water were identified using the following screening criteria;

- **EPA Region III COPC Screening Levels for Tap Water Ingestion.** Although surface water in the Area I study areas is not currently used as a drinking water supply and is not expected to be used as such in the future (Ferry Creek is tidally influenced), risk-based concentrations for tap water ingestion were used to conservatively identify COPCs. The EPA Region III criteria are calculated using an age-adjusted exposure equation, which assumes that a receptor uses a water supply for household purposes at a frequency of 350 days per year for a 30 year exposure period. The screening values for tap water ingestion, which actually incorporate exposure via inhalation of volatiles, were developed using the current EPA Region III RBC Table (EPA, 1998b). For carcinogenic chemicals, the values used for COPC screening are taken directly from the EPA Region III RBC Table and are based on a 1E-6 target incremental lifetime cancer risk. The criteria for noncarcinogenic chemicals from the EPA Region III RBC Table have been adjusted based on a target HQ of 0.1.

- **Federal and State Maximum Contaminant Levels (MCLs).** Federal MCLs are standards promulgated under the Safe Drinking Water Act and are designed for the protection of human health (direct ingestion). State MCLs have been promulgated under guidance for Connecticut agencies (Title 19, Health and Safety, the Public Code of the State of Connecticut, Chapter II Environmental Health). Both federal and state MCLs are developed in a similar manner (they are based on laboratory or

epidemiological studies and apply to drinking water supplies). They are designed in a similar manner as the EPA Region III RBCs (for the prevention of human health effects associated with lifetime exposure of an average adult, who consumes two liters of water per day). However, MCLs also reflect the technical feasibility of removing the contaminant from water. Although MCLs are typically enforceable standards for public drinking water supplies, these standards are not strictly applicable to surface waters within the Area I study areas because these waters are not currently used as a drinking water supply and they are not expected to be used as such in the future. Consequently, the use of MCLs as a COPC selection criteria is very conservative. It should also be noted that primary (health-based) MCLs are used to identify COPCs. Secondary MCLs, based on aesthetic drinking water qualities (color, odor, taste, etc.), are not used to select COPCs.

Given the relatively small size of the background and study area data sets for the Area I study areas, chemicals detected in surface water were not eliminated as COPCs on the basis of comparisons to background or on the basis of frequency of detection.

In order to identify potential ARARs exceedances, Connecticut Water Quality Standards (WQSs) (CT DEP, 1997) for the protection of saltwater and freshwater criteria and for the protection of human health were used to further evaluate chemical concentrations in the surface water in the Area I study areas (Appendix F-4), but were not used to select COPCs for quantitative risk assessment. Saltwater and freshwater criteria are considered because Ferry Creek is tidally influenced (the salinity in this resource fluctuates).

It should be noted that federal AWQCs are other health-based benchmarks that were not used to identify surface water COPCs or identify potential ARARs exceedances in the baseline human health risk assessment. These criteria were not used because they are extremely similar to the risk-based and health-based criteria identified in the previous paragraphs.

6.2.1.3 Exposure Point Concentrations

According to the regional guidance, risk assessments are conducted using an exposure point concentration for each COPC (except when assessing exposure to groundwater, where the maximum detected concentration and the average plume concentration are used as exposure point concentrations). The exposure point concentration is defined as the 95 percent upper confidence limit (UCL) and is calculated using the latest risk assessment guidance from EPA (EPA, 1992d and 1993d). A value of one-half the detection limit is substituted for nondetected values in the calculation. Because of potential problems with sample heterogeneity, the maximum detected concentration reported for field duplicate pair samples was used in the calculation for soil and sediment matrices at the direction of EPA. The average for the duplicate pair was employed for aqueous matrices.

Within Area A-I, subsets of data were used to determine exposure point concentrations based on differing exposure scenarios. These subsets are described in the area-specific section. Sample lists for each receptor evaluated are provided in Appendix F-5.

For sample sets consisting of 10 or less samples, the maximum concentrations were used as the exposure point concentrations for the reasonable maximum exposure (RME) and the average concentrations were used for the central tendency exposure (CTE), since the UCL does not provide a good estimation of the upper bound of the mean concentration for these small data sets (EPA, 1992d). For larger sample sets, the methodology used depends on the distribution of the sample set. For this risk assessment, the distribution was determined using the Shapiro-Wilk W-Test (Gilbert, 1987). When the results of the test were inconclusive and the distribution was regarded as undefined, the distribution was assumed to be log normal and the 95 percent UCL for log-normally distributed data sets was selected as the exposure point concentration for both the RME and CTE cases, unless the 95 percent UCL for log-normally distributed data sets exceeded the maximum reported concentration. In all large data sets whenever the appropriate 95 percent UCL exceeded the maximum concentration, the maximum concentration is selected as exposure point

concentration for the RME case and the average concentration is selected for the CTE case.

For normally distributed data, the calculation of the UCL is a two-step process. First the standard deviation of the sample set must be determined, as follows:

$$S = \left[\frac{\Sigma(X_i - \bar{X})^2}{(n-1)}\right]^{1/2}$$

where:
- S = standard deviation
- X_i = individual sample value
- n = number of samples
- $\bar{x}$ = mean sample value

The one-sided UCL on the mean is then calculated as follows:

$$UCL = \bar{X} + t\left(\frac{S}{n^{1/2}}\right)$$

where:
- UCL = 95 percent Upper confidence limit of the mean
- $\bar{X}$ = Arithmetic average
- t = One-sided t distribution factor ($t_{0.95, n-1}$)
- S = standard deviation
- n = number of samples

For log-normally distributed data sets, the UCL is calculated using the following equation:

$$UCL = \exp\left(\bar{X} + 0.5s^2 + \frac{Hs}{(n-1)^{1/2}}\right)$$

where:
- UCL = 95 percent UCL of the mean
- exp = Constant (base of the natural log, e)

$\bar{X}$ = Mean of the transformed data

s = Standard deviation of the transformed data

H = H-statistic (from Gilbert, 1987; $H_{0.95}$)

n = Number of samples

This equation uses individual sample results that have been transformed by taking the natural logarithm of the results.

Sample calculations for determining the distribution of a data set, UCL, and average and maximum plume concentrations are provided in Appendix F-6. After the UCL was calculated, it was compared to the maximum detected concentration within the data set; the smaller of the two was selected as the exposure point concentration for the RME case. Whenever the UCL exceeded the maximum, the average was selected as the exposure point concentration for the CTE case.

6.3 Toxicity Assessment

The toxicity assessment for the COPCs examines information concerning the potential human health effects of exposure to COPCs. The goal of the toxicity assessment is to provide, for each COPC, a quantitative estimate of the relationship between the magnitude and type of exposure and the severity or probability of human health effects. The toxicity values presented in this section are integrated with the exposure assessment (Section 6.4) to characterize the potential for the occurrence of adverse health effects (Sections 6.5 and the site-specific sections).

The toxicological evaluation involves a critical review and interpretation of toxicity data from epidemiological, clinical, animal, and in vitro studies. This review of the data determines both the nature of the health effects associated with a particular chemical, and the probability that a given quantity of a chemical could result in the referenced effect. This analysis defines the relationship between the dose received and the incidence of an adverse effect for the chemicals of potential concern.

The entire toxicological database is used to guide the derivation of cancer slope factors (CSFs) for carcinogenic effects and Reference Doses (RfDs) for noncarcinogenic effects. These data may include epidemiological studies, long-term animal bioassays, short-term tests, and evaluations of molecular structure. Data from these sources are reviewed to determine if a chemical is likely to be toxic to humans. Because of the lack of available human studies, however, the majority of toxicity data used to derive CSFs and RfDs comes from animal studies.

For noncarcinogenic effects, the most appropriate animal model (the species most biologically similar to the human) is identified. Pharmacokinetic data often enter into this determination. In the absence of sufficient data to identify the most appropriate animal model, the most sensitive species is chosen. The RfD is generally derived from the most comprehensive toxicology study that characterizes the dose-response relationship for the critical effect of the chemical. Preference is given to studies using the exposure route of concern; in the absence of such data, however, an RfD for one route of exposure may be extrapolated from data from a study that evaluated a different route of exposure. Such extrapolation must take into account pharmacokinetic and toxicological differences between the routes of exposure. Uncertainty factors are applied to the highest no-observed-adverse-effect-level (NOAEL) to adjust for inter- and intraspecies variation, deficiencies in the toxicological database, and use of subchronic rather than chronic animal studies. Additional uncertainty factors may be applied to estimate a NOAEL from a lowest-observed-adverse-effect-level (LOAEL) if the key study failed to determine a NOAEL. When chemical-specific data are not sufficient, an RfD may be derived from data for a chemical with structural and toxicologic similarity.

CSFs for weight-of-evidence Group A or B chemicals are generally derived from positive cancer studies that adequately identify the target organ in the test animal data and characterize the dose-response relationship. CSFs are derived for Group C compounds for which the data are sufficient but are not derived for Group D or E chemicals. (An explanation/definition of these weight-of-evidence classes is provided in Section 6.3.1.) No consideration is given to similarity in the animal and human target organ(s), because a

chemical capable of inducing cancer in any animal tissue is considered potentially carcinogenic to humans. Preference is given to studies using the route of exposure of concern, in which normal physiologic function was not impaired, and in which exposure occurred during most of the animal's lifetime. Exposure and pharmacokinetic considerations are used to estimate equivalent human doses for computation of the CSF. When a number of studies of similar quality are available, the data may be combined in the derivation of the CSF.

Toxicological profiles for each of the major COPCs are presented in Appendix F-7. These profiles present a summary of the available literature on carcinogenic and noncarcinogenic effects associated with human exposure to the chemical. Brief summaries of the toxicity profiles for the major COPCs are presented in Section 6.3.3

6.3.1 Carcinogenic Effects

The toxicity information considered in the assessment of potential carcinogenic risks includes a weight-of-evidence classification and a slope factor. The weight-of-evidence classification qualitatively describes the likelihood that a chemical is a human carcinogen and is based on an evaluation of the available data from human and animal studies. A chemical may be placed in one of three groups in EPA's classification system to denote its potential for carcinogenic effects:

- Group A - known human carcinogen,
- Group B1 or B2 - probable human carcinogen, and
- Group C - possible human carcinogen.

Chemicals that cannot be classified as a human carcinogen due to of a lack of data are placed in Group D, and those for which there is evidence of noncarcinogenicity in humans are in Group E.

The CSF is the toxicity value used to quantitatively express the carcinogenic hazard of cancer-causing chemicals. It is defined as the upperbound estimate of the probability of cancer incidence per unit dose averaged over a lifetime. Slope factors are derived from studies of carcinogenicity in humans and/or laboratory animals and are typically calculated for compounds in Groups A, B1, and B2, however, some Group C carcinogens also have slope factors and some B2 carcinogens, such as lead, have none. Slope factors are specific to a chemical and route of exposure and are expressed in units of $(mg/kg/day)^{-1}$ for both oral and inhalation routes. Inhalation cancer toxicity values are usually expressed as inhalation unit risks in units of reciprocal $\mu g/m^3$ $[1/(\mu g/m^3)]$. Because cancer risk characterization requires an estimate of reciprocal dose in units of 1/(mg/kg/day), the inhalation unit risk must be converted to the mathematical equivalent of an inhalation cancer slope factor, or risk per unit dose (mg/kg/day). This is done by assuming that humans weigh 70 kg and inhale 20 m^3 of air per day [i.e., the inhalation unit risk $(1/\mu g/m^3)$ is divided by 20 m^3, multiplied by 70 kg, and multiplied by 1,000 $\mu g/mg$ to yield the mathematical equivalent of an inhalation slope factor (1/mg/kg/day)]. CSFs for COPCs at the Area I study areas are presented in Table 6-3. The primary sources of information for these values are the EPA Washington (EPA, 1997b and 1998) and EPA Region III (EPA, 1998b).

EPA's database (IRIS - the Integrated Risk Information System) (EPA, 1998) was consulted as the primary source for CSF values, as well as for RfDs. EPA intends that IRIS supersede all other sources of toxicity information for risk assessment. If values are not available in IRIS, the annual Health Effects Assessment Summary Tables (HEAST) (EPA, 1997b) were consulted, as well as the current EPA Region III Risk-Based Concentration table (EPA, 1998b). If no CSF is available from any of these sources, carcinogenic risks are not quantified and potential exposures are addressed in the general uncertainty section, Section 6.6.

CSFs exist for several (but not all) Class C compounds, which are identified as "possible" human carcinogens. These compounds typically exhibit inadequate evidence of carcinogenicity in humans and limited evidence in animals. In this human health risk

assessment, Class C compounds are evaluated quantitatively as class A/B1/B2 compounds, but the risks associated with exposure to Class C compounds are also discussed separately if these chemicals are major risk drivers, underscoring the uncertainty associated with these estimations.

Dermal CSFs are derived from the corresponding oral values. In the derivation of a dermal CSF, the oral CSF is divided by the gastrointestinal absorption efficiency to determine a CSF based on an absorbed dose rather than an administered dose. The oral CSF is divided by the absorption efficiency because CSFs are expressed as reciprocal doses. Dermal CSFs and the absorption efficiencies used in their determination are also included in Table 6-3. Adjustments were made to the oral CSFs according to EPA guidance following Table 4.1, "Summary of Gastrointestinal Absorption Efficiencies and Recommendations for Adjustment of Oral Slope Factors for Specific Compounds" (EPA, 1998c).

Risk estimates for PAHs have (in the past) assumed that all carcinogenic PAHs have a potency equal to that for benzo(a)pyrene. While benzo(a)pyrene was well studied, other Class B2 PAHs had insufficient data to calculate a CSF. EPA has published provisional guidance to assess PAHs (EPA, 1993b). Estimated orders of potential potency (rather than a toxicity equivalence factor or TEF) were developed based on skin painting tests and are rounded to one significant figure (based on an order of magnitude). The values are based on a comparable endpoint (complete carcinogenesis after repeated exposure to mouse skin). The quality of the data does not support any greater precision. The orders of potential potency used in this health risk assessment are presented in Table 6-4 and are those proposed for use by EPA Region I (EPA, 1994f).

EPA has determined that the oral CSF for benzo(a)pyrene is 7.3 $(mg/kg/day)^{-1}$. A provisional inhalation CSF of 3.1 $(mg/kg/day)^{-1}$ was presented by NCEA (EPA, 1998b).

In light of the following statements from the "Dermal Risk Assessment, Interim Guidance" (EPA, 6/19/97) oral CSFs and RfDs for PAHs were used and not adjusted (EPA, 1998c) to evaluate dermal risk from PAHs: "The statement in RAGS claiming that it is inappropriate

to use the oral slope factor to evaluate the risks associated with exposure to carcinogens such as benzo(a)pyrene which causes skin cancer through direct action at the point of application should not be interpreted to mean that exposure to dermally active chemicals should not be evaluated. In fact, there is a significant body of evidence in the literature to generate a dose-response relationship for the PAH effects as a result of dermal application of PAHs to the skin surface. In addition, PAHs have also been shown to induce systemic toxicity and tumors at distant organs. For these reasons, the lack of dermal toxicity values may significantly underestimate the risk of exposure to PAHs in soil."

The toxicity and cancer risk characterization assessment for chlorinated dioxin and furan congeners is performed using TEF methodology (EPA, 1989e). The TEFs presented in Table 6-5 were used to convert concentrations of dioxin and furan congeners to individual TEQs of TCDD. The total amount of toxic dioxin and furan congeners present at a site is usually expressed as toxic equivalents (TEQ) of 2,3,7,8- tetrachlorodibenzodioxin (TCDD) present. The total TEQ of TCDD concentration is evaluated in the risk characterization to produce cancer risk estimates for exposures to chlorinated dioxin and furan congeners. Sample calculations of dioxin TEQs are presented in Appendix F-6.

The toxicity and cancer risk characterization of PCBs and dioxin-like PCB congeners was conducted according to guidance presented in the EPA technical guidance document entitled, "PCBs: Cancer Dose-Response Assessment and Application to Environmental Mixtures" (EPA, 1996e). The guidance document suggests methodology for the risk evaluation of the total Aroclor concentration in an environmental media as well as the evaluation of exposure to the dioxin-like PCB congeners that may be present. The assessment methodology uses "low" to "high risk" cancer slope factors for total Aroclors that reflect: 1) the influence on toxicity of PCBs by chemical transformation in the environment; 2) the tendency of PCBs to partition into various media; and 3) the potential for PCBs to biomagnify through the food chain. The assessment recommends a tiered approach for determining central tendency and high end cancer slope factors for use in risk assessment. When PCB congener information is limited, the exposure pathway is used to indicate whether environmental processes have increased or decreased a PCB mixture's

potency. When PCB congener information is available, further refinement of the potency estimate can occur. Three categories of slope factors were developed based on the exposure pathway or, if more information is available, the PCB congener makeup of the mixture (Table 6-6A). A "high-risk" category is used for exposure pathways associated with environmental processes that tend to increase risk; a "low-risk" category for those that tend to decrease risk; and a "lowest risk" category for cases where congener or isomer analyses verifies the absence of congeners with more than four chlorines per molecule (establishing sufficient similarity of an environmental mixture to the least potent PCB Aroclor tested). Conservatively, the "high-risk" cancer slope factor was used in this baseline risk assessment to evaluate the Aroclor data because: 1) there is limited PCB congener data available (one to two samples per study area), and 2) the PCB congener data that is available suggests that "dioxin-like" PCBs are present in the environmental media. The PCB congener data available for each site were evaluated using the toxicity equivalent factors and cancer slope factors presented in Tables 6-6A and 6-6B. The results of the toxicity assessment and cancer risk characterization of the PCB congener data are presented and discussed in the uncertainty section for each study area because the limited amount of PCB congener data cannot be used to characterize the study areas as a whole.

The cancer risk estimates presented in this assessment were based on total Aroclor concentrations. Total Aroclor concentrations were determined on a sample-specific basis by summing individual Aroclor concentrations; one-half the detection limit was used as a surrogate for non-detect results. In situations in which only one or two Aroclors are detected, the total Aroclor value may be strongly influenced by detection limits of non-detected Aroclors. For example, the only individual Aroclor detected in surface water was Aroclor 1262, detected in Area A-3 surface water. The evaluation of risks due to total Aroclor concentrations in surface water in Area A-3 results in risks that are strongly influenced by one-half detection limit values for the other Aroclors. For this reason, risks due to Aroclors in surface water have been evaluated using the Aroclor 1262 concentrations only, rather than using the artificially elevated total Aroclor concentrations.

6.3.2 Noncarcinogenic Effects

For noncarcinogens, it is assumed that there exists a dose below which no adverse health effects will be seen. Below this "threshold" dose, exposure to a chemical can be tolerated without adverse effects. For noncarcinogens, a range of exposure exists that can be tolerated. Toxic effects are manifested only when physiologic protective mechanisms are overcome by exposures to a chemical above its threshold level. Maternal and developmental endpoints are considered systemic toxicity.

The potential for noncarcinogenic health effects resulting from exposure to chemicals is assessed by comparing an exposure estimate (intake or dose) to a Reference Dose (RfD). The RfD is expressed in units of mg/kg/day and represents a daily intake of contaminant per kilogram of body weight that is not sufficient to cause the threshold effect of concern. An RfD is specific to the chemical, the route of exposure, and the duration over which the exposure occurs. Separate RfDs are presented for ingestion and inhalation pathways. In particular, Reference Concentrations (RfCs) in units of mg/m^3 are typically presented for the inhalation pathway. Because characterization of noncarcinogenic effects requires an estimate of dose in units of mg/kg/day, the inhalation RfC must be converted to an inhalation RfD. The conversion is performed by assuming that humans weigh 70 kg and inhale 20 m^3 of air per day [i.e., the inhalation RfC (mg/m^3) is multiplied by 20 m^3/day and divided by 70 kg to yield an inhalation RfD (mg/kg/day)].

To derive an RfD, EPA reviews all relevant human and animal studies for each compound and selects the study (studies) pertinent to the derivation of the specific RfD. Each study is evaluated to determine the no-observed-adverse-effect-level (NOAEL) or, if the data are inadequate for such a determination, the lowest-observed-adverse-effect-level (LOAEL). The NOAEL corresponds to the dose (in mg/kg/day) that can be administered over a lifetime without inducing observable adverse effects. The LOAEL corresponds to the lowest daily dose that induces an observable adverse effect. The toxic effect characterized by the LOAEL is referred to as the "critical effect." To derive an RfD, the NOAEL (or LOAEL) is divided by uncertainty factors to ensure that the RfD will be

protective of human health. Uncertainty factors are applied to account for extrapolation of data from laboratory animals to humans (interspecies extrapolation), variation in human sensitivity to the toxic effects of a compound (intraspecies differences), derivation of a chronic RfD based on a subchronic rather than a chronic study, or derivation of an RfD from the LOAEL rather than the NOAEL. In addition to these uncertainty factors, modifying factors between one and 10 may be applied to reflect additional qualitative considerations in evaluating the data. For most compounds, the modifying factor is one.

A dermal RfD is developed by multiplying an oral RfD (based on an administered dose) by the gastrointestinal tract absorption factor. The resulting dermal RfD, based on an absorbed dose, is used to evaluate the dermal (absorbed) dose calculated by the dermal exposure algorithms.

Reference Doses for the COPCs at the Area I study areas are presented in Table 6-7. The primary source of these values is the IRIS database, followed by other EPA sources described for the carcinogens. This table also includes the primary target organs affected by a particular chemical. This information may be used in the Risk Characterization section to segregate risks by target organ effects, unless the total Hazard Index is below unity.

As discussed above, PCB risk characterization is generally addressed by evaluation of total Aroclor concentrations. For non-carcinogenic risk, however, two PCB congeners, Aroclor 1016 and Aroclor 1254, have oral RfDs available and a noncarcinogenic risk evaluation can be performed. The oral RfD for Aroclor 1016 is 7.00E-05 mg/kg/day and the oral RfD for Aroclor 1254 is 2.00E-05 mg/kg/day. Noncancer risk estimates for these two Aroclors are presented in this assessment.

6.3.3 Toxicity Summaries for Major Chemicals of Concern

This section contains brief summaries of the toxicological profiles for the major COPCs. The detailed profiles are contained in Appendix F-7.

6.3.3.1 Polyaromatic Hydrocarbons (PAHs)

Benzo(a)pyrene is the most widely studied chemical in this class. It is used as the basis for defining the toxicity of other potentially carcinogenic PAHs. Benzo(a)pyrene is widely distributed in the tissues of treated rats and mice but is primarily found in tissues high in fat. While the carcinogenicity of complex mixtures containing PAHs (such as coal tar, coke oven emissions, and cigarette smoke) is suggested, the carcinogenicity cannot be attributed solely to PAHs. The carcinogenicity of benzo(a)pyrene is based largely on the results of animal studies in which the animals were exposed to large doses of purified compound via atypical routes of exposure.

The noncarcinogenic PAHs appear to affect the liver, kidneys, and blood of exposed laboratory animals. Considered exposure routes include ingestion and inhalation, and exposure has resulted in anemia and mild liver lesions and occasionally renal disease. The effects vary for the individual compounds.

6.3.3.2 Lead

Unborn children and young children are particularly sensitive to the adverse effects of exposure to lead. Exposure to a fetus through its mother may cause premature births, lower birth weight, and decreased mental ability of the infant. Lead exposure is dangerous for young children because they absorb lead at a greater rate than adults, retain more of the lead they ingest, and are more sensitive to its effects. Effects include decreased intelligence and decreased growth.

Lead is efficiently absorbed by children. The fate of lead in the body depends in part on the amount and rate of previous exposures, the age of the receptor, and the rate of exposure. The principal effects of acute oral exposure are colic, anemia, and, in severe cases, acute encephalopathy (particularly in children). Long-term exposure may result in neurological and hematological effects. Some of the effects on the blood and subtle neurobehavioral changes in children occur at levels so low that they are considered

nonthreshold effects. Rat and mouse studies have shown increases in renal tumors, but the human studies have yielded inconclusive results that failed to account for the presence of other potentially carcinogenic materials. EPA has classified lead as a B2 carcinogen based on the results of animal studies.

6.3.3.3 Copper

A deficiency of copper, an essential element, may result in anemia, loss of pigment, reduced growth, and loss of arterial elasticity. However, persons who are overexposed may exhibit Wilson's disease (disorder of copper metabolism) or liver cirrhosis (Lappenbusch, 1988).

6.3.3.4 Barium

Increased blood pressure has been observed in experimental animals (rats) routinely exposed to barium in drinking water. Barium is also toxic to the nervous system, the muscular system, and gastrointestinal system when ingested at high concentrations. The soluble barium salts are more toxic than the insoluble barium salts (Clements Associates Inc., 1985). This is probably due to the fact that the soluble barium salts are more likely to be absorbed than the insoluble barium salts.

6.3.3.5 Polychlorinated Biphenyls (PCBs)

PCBs are a group of synthetic organic chemicals that contain 209 individual compounds (known as congeners). Mixtures of PCBs, or Aroclors, were manufactured for use in industry until 1977. EPA considers PCBs to be a probable human carcinogen based on evidence of the ability of PCBs to cause cancer in animal studies. Data on humans exposed to PCBs suggest an association between PCB exposure and human cancer, but lack of data on exposure dose, length of exposure, types of PCBs and other chemicals people were exposed to precludes identification of a cause and effect relationship based only on human studies. Studies in animals have also demonstrated immunological,

reproductive, and neurological effects from PCB exposure. Studies in mice, monkeys, guinea pigs, and rabbits have shown PCBs to be immunosuppressive. Some PCB congeners are considered dioxin-like.

6.3.3.6 Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurans

Polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are chemically classified as halogenated aromatic hydrocarbons. The most widely studied of these compounds is 2,3,7,8 tetrachlorodibenzo-*p*-dioxin. This compound, often called simply dioxin, represents the reference compound for this class of compounds. Toxic Equivalency Factors (TEFs) are used to estimate the toxicity of other PCDDs and PCDFs which have chlorine in the 2,3,7, and 8 position. These compounds have been widely publicized as the most potent man-made toxicants ever studied. Exposure to dioxin and related compounds is associated with subtle biochemical and biological changes and with chloracne, a serious skin condition. Laboratory studies suggest the probability that exposure to dioxin-like compounds may be associated with other health effects including cancer. Dioxins have been demonstrated to be potent modulators of cellular growth and differentiation, particularly in epithelial tissues.

6.3.3.7 Asbestos

Asbestos is poorly absorbed from the gastrointestinal tract and therefore displays low acute oral toxicity. However, respiratory exposure leads to pulmonary fibrosis called asbestosis, which symptoms include breathlessness, chest pain, cough, decreased lung function, and cyanosis. Occupational exposures to asbestos have resulted in higher incidences of lung cancer, especially in combination with cigarette smoking; the latent period is 15 to 30 years. An additional effect of asbestos exposure is the development of pleural or peritoneal mesotheliomas; the latent period is 3.5 to 30 years (Hodgson et al., 1988).

6.4 Exposure Assessment

The exposure assessment defines and evaluates the exposures experienced by a receptor population. In order to have an exposure, several factors must be present: first, there must be a source of contamination; second, there must be a mechanism through which a receptor can come into contact with the contaminants in that medium; and third, there must actually (or potentially) be a receptor present at the point of contact.

The exposure assessment presented in this section of the report consists of several sections that characterize the physical site setting and the receptors of concern, identify the potential contaminant migration and exposure pathways, define the contaminant concentrations at the point of exposure, and present the equations used to quantify exposure in terms of contaminant intake (dose). Appendix F-8 of this report contains sample calculations for the exposure assessment. Tables of intakes are not presented in the body of the report, but the calculated values may be seen within the site-specific spreadsheets in Appendix F-9 through Appendix F-11.

6.4.1 Exposure Setting

This section contains information on the land use and receptor characteristics in the Area I study areas.

Land/Water Use. The Area I study areas were described in detail in Section 2.0 and are shown in Figure 1-2. Summarizing, the study areas include: several commercial/industrial properties and Ferry Creek and other ecological areas (the delineated wetland boundaries along Ferry Creek) impacted by Raymark Facility waste. Property within the Area I study areas has been developed for commercial/industrial purposes or is undeveloped (wetlands). None of the property within the Area I study areas has been developed for residential purposes. The physical conditions within much of the study areas (wetlands) would limit or preclude residential development. However, residential areas do border the Area I study areas.

The lower reach of Ferry Creek and the Housatonic River are used for recreational fishing and boating. Evaluation of biota will be included in the RI for the Area II study areas.

Study-area specific land use and site access information is presented in Sections 6.7 through 6.9.

Exposed Populations. The Area I study areas are located in Stratford, Fairfield County, Connecticut. The principal industries within the community of Stratford include manufacturing of aircraft, air conditioning, brake linings, chemicals, plastic, paper, rubber goods, electrical and machine parts, and toys. There were 49,389 people reported on the 1990 census for the Town of Stratford. The Stratford Town Clerk reported this as a slight decrease from the last census in 1980. Potentially exposed populations within each Ferry Creek study area are discussed in Sections 6.7 through 6.9.

6.4.2 Conceptual Site Model

This section discusses the general conceptual site model for the Area I study areas. A conceptual site model facilitates consistent and comprehensive evaluation of the risks to human and ecological health by creating a framework for identifying the paths by which human health may be impacted by contaminants predicted to exist at the source areas. A conceptual site model depicts the relationships between the elements necessary to construct a complete exposure pathway, as follows;

- Sources and potential COPCs
- Contaminant release mechanisms
- Contaminant transport pathways
- Exposure mechanisms and exposure routes
- Receptors

One simple conceptual site model was developed for all study areas to provide the basis for identifying the potential risks to human health and the environment. The model

considers the current and future conditions within the study areas and the actual or potential receptors who could come into contact with the COPCs.

The conceptual site model first considers the contaminant sources assumed to be available, either currently or in the future. The sources are Raymark Facility soil-waste migrating from the Raymark Facility or disposed within the study areas, or the contaminated soils, wetland soils, and sediments within the study areas. Contaminants may be released from these sources by mechanisms such as wind or water erosion or leaching to the subsurface. Once released from a source, contaminants are transported in media such as air, surface water, or groundwater. Receptors may be exposed either directly or indirectly to contaminants in environmental media via a variety of mechanisms. The exposure mechanisms considered include recreational activities, working outdoors, etc. These exposure mechanisms generally act along one or more exposure routes such as ingestion, inhalation, or direct dermal contact.

The conceptual site models also indicate those exposure routes that are carried through the quantitative risk assessment for each receptor. An objective of the development of the conceptual site model is to focus attention on those pathways that contribute the most to the potential impacts on human health and the environment, and to provide the rationale for screening out other exposure pathways that are minor components of the overall risk.

6.4.2.1 Sources of Contamination

The Raymark Industries, Inc. (Raymark) facility, formerly named Raybestos - Manhattan Company, is located at 75 East Main Street in Stratford, Fairfield County, Connecticut. This facility occupied 33 acres and manufactured friction materials containing asbestos and non-asbestos materials, metals, phenol-formaldehyde resins, and various adhesives. Primary products were gasket material, sheet packing and friction materials including clutch facings, transmission plates, and brake linings. As a result of these activities, soils at the Raymark Facility have been contaminated primarily with asbestos, lead, and PCBs. Presently, the Raymark Industries, Inc. Facility is undergoing a source control remedial

action (Operable Unit No. 1), consistent with the Record of Decision (ROD) signed on July 3, 1995.

Raymark operated from 1919 until 1989, when the plant was shut down and permanently closed. During Raymark's 70 years of operation, it was common practice to dispose of manufacturing waste at locations in Stratford. A number of these "locations" were the subject of time-critical removal actions conducted by the EPA and its contractors. The removal actions have identified the locations around town with the highest levels of asbestos, lead, and PCBs. Contaminants present in these areas have been designated a health threat and have been excavated, covered, and/or fenced. Other "locations" have been identified as contaminated with asbestos, lead, and PCBs. Some of these locations have been sampled and are included in this Area I study area RI.

6.4.2.2 Contaminant Release and Migration Mechanisms

Chemicals may have been released from the former Raymark Facility and from properties within the Area I study area by a variety of mechanisms including stormwater runoff and subsequent erosion of surface soil, infiltration of soluble chemicals and subsequent migration through the subsurface soil to the water table where the chemicals may migrate downgradient, and via wind erosion of surface soil from unpaved areas (Section 5.1). The Raymark Facility has been capped, therefore the current transport mechanisms from the Raymark Facility are limited to only the groundwater that migrates below the cap.

Transport mechanisms associated with storm water run-off and subsequent erosion of surface soil and wind erosion are valid within the Area I study areas. Storms generate runoff, which is directed toward stormwater drainageways. Initially, this water may move across an area as sheet flow, which can entrain loose soil material. This soil is moved as a sediment and will be deposited where the flow velocity diminishes below that needed to carry a particular grain size. Typically in undeveloped areas, this soil/sediment is deposited in small drainageways and migrates farther downstream with each new storm, which also

adds new material. Within the Area I study areas, contaminants entrained in/dissolved in surface water have migrated to Ferry Creek and bordering wetland areas.

Soluble chemicals released to the ground surface may also migrate downward through the soil column with infiltrating precipitation. The migration of these chemicals may be somewhat impeded by the chemical's tendency to bind to soil organic material. Eventually, these soluble chemicals may reach the water table. Once in the groundwater, chemicals may continue to migrate via dispersion and advection in the downgradient direction. Eventually, these chemicals may discharge with the groundwater to surface bodies such as Ferry Creek and wetlands.

Chemicals adsorbed to surface soil may also be released from a site via wind erosion of loose soil material. These particulates are carried downwind and potentially off site if the grain size is small enough and the wind velocity is great enough. Additionally, chemicals may also be released from soil via volatilization.

6.4.3 Potential Routes of Exposure

A receptor can come into contact with contaminants in a variety of ways, which are generally the result of interactions between a receptor's behavior or lifestyle and an exposure medium. This assessment defines an exposure route as a stylized description of the behavior that brings a receptor into contact with a contaminated medium.

6.4.3.1 Air

This pathway is based on the scenario that a receptor is immersed in air that contains suspended particulates and volatile organic vapors originating from the source areas as part of daily living. Subsequent exposure of the receptor occurs upon inhalation of the ambient air.

Initially, a qualitative comparison of maximum detected soil concentrations and EPA Generic SSLs for inhalation, based on intermedia transfer (from soil to air), was performed to determine if additional quantitative analysis of this potential exposure pathway was warranted. The inhalation SSLs are based on residential land use and lifetime exposure scenarios and are therefore relatively conservative values for potential receptors under current land use conditions. Exposures to fugitive dust and VOCs released from soil (shallow soil and "all soil") were found to be insignificant in most cases based on the qualitative screening, which is summarized in the site-specific COPC screening tables. Maximum chemical detections in soil were less than the SSLs for most COPCs identified in the study areas; the inhalation exposure pathway therefore was not considered for further evaluation. A discussion of the inhalation pathway, as it pertains to each of the study areas, is provided in the site-specific exposure assessments in Sections 6.7 through 6.9.

6.4.3.2 Direct Contact with Soil/Wetland Soils/Sediment

Receptors may come into direct contact with soil/wetland soils/sediments affected by the release of chemicals from the source areas. During the receptor's period of contact, the individual may be exposed via inadvertent ingestion of a small amount of soil or via dermal absorption of certain contaminants from the soil.

Because of the limited guidance available to estimate exposure to soil via dermal contact, EPA Region I recommends performing a quantitative evaluation of dermal risks for arsenic; cadmium; chlordane; lindane; 2,4-dichlorophenoxyacetic acid (2,4-D); dichlorodiphenyltrichloroethane (DDT); dioxins, PAHs (benzo(a)pyrene); PCBs (Aroclor 1254 and 1242); and pentachlorophenol, only. Most of these chemicals were selected as COPCs for the Area I study areas. Therefore, dermal risks associated with soil were quantitatively addressed in the risk assessment. Dermal contact with other chemicals detected in the site soils may or may not result in a significant exposure. It should be noted that organics such as PAHs, which were detected frequently in the soil samples and selected as COPCs, tend to strongly adhere to organic matter in soil. For these chemicals to be percutaneously absorbed, they must first desorb from soil and diffuse through the

skin. Various factors affect the rate of dermal absorption, including the amount of soil on the skin surface, soil characteristics (moisture, pH, organic carbon content, etc.), skin characteristics (thickness, temperature, hydration, etc.), volatilization losses, and chemical-specific properties.

6.4.3.3 Direct Contact with Groundwater

As discussed previously, the groundwater resource at the Area I study area is not evaluated in this baseline risk assessment. It is possible that an excavation (for construction, utility maintenance, etc.) could be deep enough to come into contact with the shallow groundwater. In such an instance, workers could be exposed to the groundwater via dermal contact. However, the nature of the Ferry Creek wetland material and local construction practices would preclude excavation/construction in much of the study areas. Potable use of groundwater is not considered to be likely to occur under current and/or future land use because of the brackish conditions.

6.4.3.4 Direct Contact with Surface Water

Receptors may also come into direct contact with surface water containing chemicals in a suspended or dissolved phase. This exposure would be of short duration and individuals may be exposed via dermal contact.

6.4.4 Potential Receptors

Several potential receptors have been identified under both current and future land use conditions. These receptors were identified by analyzing the interaction of current and anticipated future land use practices and the identified sources of contamination.

Several receptor groups have been defined for this risk assessment. These receptors are as follows;

- Commercial Workers - Adults working 40 hours per week at a commercial facility within the Area I study areas.

- Frequent recreational users - Residents (adults and children) who reside at properties located in the vicinity of a study area and who may periodically visit (recreate) within a study area.

- Trespasser - Adolescents who may occasionally trespass into a study area.

One or more of these receptor groups are evaluated quantitatively for each of the study areas under investigation in this report. Table 6-8 contains a matrix summary of the particular combinations of receptor groups developed for the Area I study areas. Figure 6-1 presents the locations of exposures evaluated for each receptor in this report. The rationale for the selection of receptor groups for each study area is provided in the study area - specific exposure assessments in Sections 6.7 through 6.9.

The adult commercial worker was evaluated for exposures to surface (depths of 0 to 2 feet bgs without pavement) and "all soils" (to a depth of 15 feet bgs regardless of pavement) for the current and future land use scenarios, respectively. Workers are not expected to contact surface water, sediment, or wetland soils.

Adult and child recreational users and adolescent trespassers were evaluated for exposures to exposed shallow soils, wetland soils, sediments, and/or surface waters. The frequent recreational user was evaluated if residential properties border a study area. The "attractiveness" of a surface water body within a study area was considered when evaluating the frequent recreational user and the potential for exposure to surface water.

Two bounding estimates of each exposure scenario are considered, as per EPA Region I guidance. The first is identified as a central tendency exposure (CTE) receptor, which was developed using both regional guidance (EPA, 1994f) and professional judgment regarding

site-specific conditions. The second class of receptor is called the reasonable maximum exposure (RME) and was developed according to EPA guidance (EPA, 1989d and 1994f).

6.4.5 Exposure Pathways

An exposure pathway consists of four elements: a source and mechanism of release, a route of contaminant transport through an environmental medium, a contact point for a human receptor, and an exposure route at the point of contact. All four components must be present for the exposure pathway to be considered complete. This section summarizes the potentially complete exposure pathways that are quantitatively evaluated in the risk assessment and provides the rationale for those pathways that are not evaluated. Table 6-9 presents a summary of the potentially complete and incomplete exposure pathways and receptors.

6.4.6 Quantification of Exposure

Estimates of exposure are based on the contaminant concentrations at the exposure points and on scenario-specific assumptions and intake parameters. The models and equations used to quantify intakes are described in this section and have been obtained from a variety of EPA guidance documents, which are cited in the specific intake estimation sections that follow.

Exposures depend on the predicted concentrations of chemicals in environmental media and local land use practices, and both are subject to change over time. This results in a large number of possible combinations of receptors, media, exposure pathways, and concentrations. As mentioned previously, Table 6-9 presents a summary of the exposure pathways evaluated in the quantitative risk assessment. Some of these scenarios (such as occupational, trespassing, and recreational scenarios) may be applicable under both current and future land use conditions.

Exposure model parameters are presented in Tables 6-10 and 6-11 for soils/sediments and surface water, respectively. The values reflect current EPA guidance and comments received from EPA Region I on the Draft Final Work Plan Remedial Investigation/Feasibility Study for the Raymark-Ferry Creek study areas, December, 1996. All parameters are referenced in footnotes on each table. These parameters are used in the equations presented in this section, along with the exposure point concentrations presented in the site-specific sections, to calculate intakes, which are used to determine risks. Individual chemical intakes for each receptor/exposure route combination are presented in the spreadsheets in Appendices F-9 through F-11.

Incidental Ingestion of Soil/Wetland Soil/Sediment. The estimation of intake of contaminants in soils/sediments is determined using the exposure point concentration of a contaminant in the study area of interest. This pathway is evaluated for both child and adult receptors involved in recreational activities, commercial workers, and adolescent trespassers. In general, intakes associated with soil ingestion are calculated using the following equation:

$$Intake_{si} = \frac{(C_{si})(IR)(FI)(EF)(ED)(CF)}{(BW)(AT)}$$

where: $Intake_{si}$ = intake of contaminant "i" from soil (mg/kg/day)

C_{si} = concentration of contaminant "i" in soil (mg/kg)

IR = ingestion rate (mg/day)

FI = fraction ingested from contaminated source (decimal fraction)

EF = exposure frequency (days/yr)

ED = exposure duration (yr)

CF = conversion factor (10-6 kg/mg)

BW = body weight (kg)

AT = averaging time (days);
for noncarcinogens, AT = ED*365 days/yr;
for carcinogens, AT = 70 yr*365 days/yr

To evaluate the RME, TtNUS used soil ingestion rates of 100 mg/day for the commercial/industrial worker under current and future land use, 200 mg/day for child recreational users, and 100 mg/day for adolescent trespassers and adult recreational users. Soil ingestion rates for the CTE were set at 100 mg/day for child recreational users and 50 mg/day for adolescent trespassers, adult recreational users, and commercial/industrial workers.

No attempt was made to vary a receptor's exposure frequency for the RME and CTE. A value of 250 days/year was used for workers which is consistent with EPA and DEP default values. Site-specific considerations were used to determine exposure frequencies for the remaining potential receptors. A value of 150 days/year was used for frequent recreational users for Areas A-1 and A-2. This value assumes that the receptor is exposed approximately three days/week year-round. For Area A-3, an exposure frequency of 90 days/year was used for frequent recreational users because the wet nature of this area was assumed to limit visits to the period of April through October. Trespassing is expected to occur at a frequency of one day/week year-round, for a total of 52 days/year.

A majority of the proposed exposure duration values are based on EPA guidance for RME and CTE evaluation. Values for small children and older child trespassers for the RME reflect the entire age span for the receptor evaluated. The associated CTE values reflect a short period of time (basically half of the RME value). Exposure duration for commercial/industrial workers is assumed to be 25 years for the RME and nine years for the CTE. RME exposure durations for child and adult receptors under recreational scenarios are six years and 24 years, respectively. CTE exposure duration for these receptors are two years (child) and seven years (adult). In Area A-3, because of the nature of the area, young children were presumed to be three to six year olds, rather than the zero to six year olds evaluated elsewhere. For this reason exposure durations for young children in Area A-3 were set at three years (both RME and CTE). For the adolescent trespasser (ages nine to 18), exposure durations are specified as 10 years for the RME and five years for the CTE.

Table 6-10A contains a summary of the input parameters for incidental ingestion of soil/sediment.

Dermal Contact with Soil/Wetland Soil/Sediment

Dermal contact exposures to soil/sediment may also occur during recreational, trespassing, and commercial/industrial scenarios. A quantitative evaluation of dermal exposure to soil/sediment was performed for arsenic, cadmium, chlordane, DDT, dioxins, PAHs, and PCBs only. Exposure to other chemicals detected at the Area I study area is addressed in a qualitative fashion.

The following equation was used to estimate the dermal adsorbed dose for soil/sediment:

$$DAD_{si} = \frac{(C)(ABS)(AF)(SA)(EF)(ED)(CF)}{(BW)(AT)}$$

where:
- DAD_{si} = Dermally absorbed dose of chemical "i" from soil/sediment (mg/kg/day)
- C = Exposure concentration for soil/sediment (mg/kg)
- ABS = Absorption factor (unitless)
- AF = Soil-to skin adherence factor (mg/cm^2)
- SA = Skin area available for contact (cm^2/day)
- EF = Exposure frequency (days/year)
- ED = Exposure duration (years)
- CF = Conversion factor (1E-6 kg/mg)
- BW = Body weight (kg)
- AT = Averaging (days);
  - AT = 70 years * 365 days/year for carcinogens;
  - AT = ED * 365 days/year for noncarcinogens

Chemical-specific absorption factors (ABS) presented in current dermal assessment guidance (EPA, 1998c) or in the literature as suggested by EPA Region I were used to

estimate absorbed doses. Unfortunately, limited information regarding dermal absorption is available. The cited guidance presents sufficient data to evaluate arsenic, cadmium, chlordane, lindane, 2,4-dichlorophenoxyacetic acid (2,4-D), dichlorodiphenyltrichloroethane (DDT), dioxins, PAHs (benzo(a)pyrene), PCBs (Aroclor 1254 and 1242), and pentachlorophenol, only. The ABS for benzo(a)pyrene, was applied as a surrogate for all PAHs and the ABS for Aroclor 1242 and Aroclor 1254 was applied as a surrogate for all PCBs. The ABS ranges presented for these chemicals are presented in Table 6-10B. Because of the absence of dermal absorption data, TtNUS qualitatively evaluated dermal exposures to all other COPCs.

Values of 0.07 mg/cm^2 and 0.01 mg/cm^2 were used as soil-to-skin adherence factors for the RME and CTE, respectively, for adult recreational users and adolescent trespassers in dry or predominantly dry areas (Areas A-1 and A-2). Values of 0.2 mg/cm^2 and 0.06 mg/cm^2 were used as soil-to-skin adherence values for the RME and CTE, respectively for child recreational users in Area A-1. For Area A-3, which is a very wet area, soil-to-skin adherence factors of 0.3 mg/cm^2 and 0.04 mg/cm^2 were used for adult recreational RME and CTE cases, respectively; and soil-to-skin adherence factors of 1.0 mg/cm^2 and 0.2 mg/cm^2 were used for the child recreational RME and CTE cases, respectively. Soil-to-skin adherence factors of 0.2 mg/cm^2 and 0.02 mg/cm^2 were used for the commercial worker for the RME and CTE cases, respectively. The values have been recommended in working drafts of EPA Interim Guidance entitled: "Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual, Supplemental Guidance, Dermal Risk Interim Guidance"(EPA, 1998c). The values were based on data presented in the 1997 version of the EPA Exposure Factor Handbook.

For the skin surface areas available for contact, the proposed values were determined by making assumptions about a receptor's exposed body areas. For adult commercial workers, the face, hands, and forearms are expected to be available for contact. For adult recreational and adolescent receptors, the face, hands, forearms, and lower legs are expected to be available for contact. For child receptors, the face, hands, forearms, lower legs, and feet are expected to be available for dermal contact. Skin surface areas

corresponding to each of these areas of the body for each age group were obtained from the Exposure Factors Handbook (EPA, 1997).

Exposure frequencies and exposure duration for dermal contact are the same as those previously identified for incidental ingestion of soil/sediment.

Input parameters for dermal contact with soil/sediment are summarized in Table 6-10A.

Inhalation of Fugitive Dust and Volatile Emission

As discussed previously, a qualitative evaluation (comparison of study area data to EPA SSLs for transfers from soil to air) of this exposure pathway was performed for each study area. No quantitative evaluation was conducted because no significant exceedances of EPA SSLs were observed.

Dermal Contact with Surface Water

Adolescent trespassers and recreational users were evaluated for dermal exposure to surface water while wading. The following equation was used to estimate exposures resulting from dermal contact with water (EPA, 1992b):

$$DAD_{wi} = \frac{(DA_{event})(EV)(ED)(EF)(SA)}{(BW)(AT)}$$

where:

DAD_{wi}	=	dermally absorbed dose of chemical "i" from water (mg/kg/day)
DA_{event}	=	absorbed dose per event (mg/cm^2-event)
EV	=	event frequency (events/day)
ED	=	exposure duration (yr)
EF	=	exposure frequency (days/yr)
SA	=	skin surface area available for contact (cm^2)
BW	=	body weight (kg)

AT = averaging time (days);
for noncarcinogens, AT = ED*365 days/yr;
for carcinogens, AT = 70yr*365 days/yr

The absorbed dose per event (DA_{event}) is estimated using a nonsteady-state approach for organic compounds and a more traditional steady-state approach for inorganics. For organics, the following equations apply:

$$\text{If } t_{event} \le t^*, \text{ then: } DA_{event} = (2K_p)(C_{wi})(CF)\left(\frac{\sqrt{6\tau t_{event}}}{\pi}\right)$$

$$\text{If } t_{event} > t^*, \text{ then: } DA_{event} = (K_p)(C_{wi})(CF)\left(\frac{t_{event}}{1+B} + 2\left(\frac{1+3B+3B^2}{(1+B)^2}\right)\right)$$

where:
t_{event} = duration of event (hr/event)
t^* = time it takes to reach steady-state conditions (hr)
K_p = permeability coefficient from water through skin (cm/hr)
C_{wi} = concentration of chemical "i" in water (mg/L)
τ = lag time (hr)
π = constant (unitless; equal to 3.141592654)
CF = conversion factor (10^{-3} L/cm^3)
B = partitioning constant derived by Bunge Model (dimensionless)

Values for the chemical-specific parameters (t_{event}, t^*, K_p, τ, and B) were obtained from the November 1998 draft dermal guidance. If no published values were available for a particular organic compound, they were calculated using equations provided in the cited guidance. Details regarding the procedures used to derive the constants, as well as sample calculations, are provided in Appendix F-8.

The following nonsteady-state equation is used to estimate DA_{event} for inorganics:

$$DA_{event} = (K_p)(C_{wi})(t_{event})(CF)$$

In general, the recommended default value of 0.001 was used for inorganic constituents.

For all receptors under the RME and CTE, the event frequency was set at one event/day. No attempt was made to vary exposure time for the RME and CTE. The exposure times for adult recreational users, child recreational users (three to six years of age), and adolescent trespassers were all set at one hour/day.

Site-specific considerations were used to determine exposure frequencies. A value of 90 days/year was used for frequent recreational users. This value assumes that the receptor is exposed approximately three days/week April through October. Trespassing is expected to occur at a frequency of one day/year, year-round.

A majority of the proposed exposure duration values are based on EPA guidance for RME and CTE evaluation. Values for small children and older child trespassers for the RME reflect the entire age span for the receptor evaluated. The associated CTE value for trespassers reflects a short period of time. RME exposure durations for child and adult receptors under recreational scenarios are three years and 24 years, respectively. CTE exposure duration for these receptors are three years (child) and seven years (adult). For the adolescent trespasser (ages nine to 18), exposure durations are specified as 10 years for the RME and five years for the CTE.

A receptor's hands, feet, and lower legs only were assumed to be exposed for the wading scenario. The skin surface areas for the wading exposure scenario for adolescent trespassers were set at 4,500 cm^2 for RME and 3,900 cm^2 for CTE. Available skin surface areas for wading for small children were assumed to be 2,300 cm^2 for RME and 2,000 cm^2 for CTE. The adult skin surface area assumed available for contact was 6,600 cm^2 for RME and 5,700 cm^2 for CTE. These values represent 90^{th} and 50^{th} percentile values for the appropriate body parts and ages of receptors as presented in the Exposure Factors Handbook, EPA, 1997.

Input parameters for dermal contact with surface water are summarized in Table 6-11.

6.4.7 Exposure to Lead

Exposure to lead was evaluated using the EPA Integrated Exposure Uptake Biokinetic (IEUBK) Model for lead, version 0.99D (EPA, 1994c). This model is designed to estimate blood levels of lead in children (under seven years of age) based on either default or site-specific input values for air, drinking water, diet, dust, and soil exposure. Exposures to lead by nonresidential adults are evaluated by use of a slope-factor approach developed by the EPA Technical Review Workgroup for Lead (EPA, 1996f and 1996g). The approach focuses on estimating fetal blood lead concentrations in women exposed to lead contaminated soils.

Blood lead concentration is the most widely used index of internal lead body burdens associated with potential adverse health effects. Studies indicate that infants and young children are extremely susceptible to adverse effects from exposure to lead. Considerable behavioral and developmental impairments have been noted in children with elevated blood lead levels. The threshold for toxic effects to children from this chemical is believed to be in the range of 10 µg/dL to 15 µg/dL. Blood lead levels greater than 10 µg/dL are considered to be a "concern."

In general, the IEUBK Model and Technical Review Work Group Model for lead were used to address exposure to lead when detected soil concentrations exceeded the OSWER soil screening level of 400 mg/kg for residential land use (EPA, 1994e). Exposure concentrations, as well as default parameters for some input parameters, were used in the evaluation. In the IEUBK model, lead concentrations in house dust were set equal to 70 percent of the lead concentration in outdoor soil, assuming a multi-source model. The IEUBK model presents 70 percent as a default value for house dust concentrations when contaminated soil is at some distance from the house and no other source of lead is known. Exposures to lead are discussed in the site-specific sections (Sections 6.7 through

6.9). The input parameters used and the results of lead models, estimated blood lead levels, and probability density histograms are presented in Appendix F-12.

6.5 Risk Characterization

This section provides a characterization of the potential human health risks associated with the potential exposure to COPCs in environmental media in the Area I study areas. Section 6.5.1 outlines the methods used to estimate the type and magnitude of health risks, and study area-specific sections (Sections 6.7 through 6.9) present the results for the current and potential future land use conditions for the Area I study areas.

6.5.1 Risk Characterization Methodology

Potential human health risks resulting from exposure to COPCs are estimated using algorithms established by EPA. The methods described by EPA are protective of human health and are likely to overestimate (rather than underestimate) risk. The methodology uses specific algorithms to calculate risk as a function of chemical concentration, human exposure parameters, and toxicity.

Risks from hazardous chemicals are calculated for either carcinogenic or noncarcinogenic effects. Some carcinogenic chemicals may also exhibit noncarcinogenic effects. Potential impacts are then characterized for both types of health effects.

Chemical Carcinogens. Risks attributable to exposure to chemical carcinogens are estimated as the probability of an individual developing cancer over a lifetime as a result of exposure to a potential carcinogen. At low doses, the incremental lifetime cancer risk (ILCR) is determined as follows (EPA, 1989d);

$$ILCR_i = (Intake_i)(CSF_i)$$

where: $ILCR_i$ = Incremental Lifetime Cancer Risk for chemical "i", expressed as a unitless probability

$Intake_i$ = Intake of chemical "i" (mg/kg/day)

CSF_i = Cancer Slope Factor of chemical "i" $(mg/kg/day)^{-1}$

Risks below 1E-6 (or a risk less than one in one million) are generally considered to be acceptable by EPA, and risks greater than 1E-4 (1 in 10,000) are generally considered to be unacceptable.

Risks are estimated for all carcinogenic compounds regardless of the class designation (See Section 6.3.1).

Noncarcinogens. The hazards associated with the effects of noncarcinogenic chemicals are evaluated by comparing an exposure level or intake to a Reference Dose (RfD). The ratio of the intake to the RfD is called the Hazard Quotient (HQ) and is defined as follows (EPA, 1989d);

$$HQ_i = \frac{Intake_i}{RfD_i}$$

where: HQ_i = Hazard Quotient for chemical "i" (unitless)

$Intake_i$ = Intake of chemical "i" (mg/kg/day), a function of exposure and chemical concentration

RfD_i = Reference Dose of chemical "i" (mg/kg/day)

If the ratio of the intake to the RfD exceeds unity, there exists a potential for noncarcinogenic (toxic) effects to occur. A Hazard Index (HI) is generated by summing the individual HQs for all the COPCs. If the value of the HI exceeds unity, it is necessary to segregate the HQs by target organ effects and access a HI for the specific target organ. If the endpoint specific HI exceeds unity, there is a potential for non-carcinogenic health effects. The HQ should not be construed as a probability in the manner of the ILCR, but

rather as a numerical indicator of the extent to which a predicted intake exceeds or is less than a RfD.

6.6 Uncertainties Analysis

There is uncertainty associated with all aspects of the baseline human health risk assessment presented in the preceding sections. This section presents a summary of these uncertainties, with a discussion of how they may affect the final risk numbers discussed in Sections 6.7 through 6.9.

There is uncertainty associated with all steps of the risk assessment process. The selection of contaminants of concern is based on exposure assumptions and toxicity information which in turn have associated uncertainties. Uncertainty in the selection of COPCs is associated with the current status of the predictive databases and the procedures used to include or exclude constituents as chemicals of concern. Uncertainty associated with the exposure assessment includes the values used as input variables for a given intake route, the methods used and the assumptions made to determine exposure point concentrations, and the predictions regarding future land use and population characteristics. Uncertainty in the toxicity assessment includes the quality of the existing data to support dose-response relationships and the weight-of-evidence used for determining the carcinogenicity of chemicals of concern. Uncertainty in risk characterization includes that associated with exposure to multiple chemicals and the cumulative uncertainty from combining conservative assumptions made in earlier activities.

While there are various sources of uncertainty, as described above; throughout the entire risk assessment, assumptions were made so that the final calculated risks would be conservative estimates which are protective of public health.

Generally, risk assessments carry two types of uncertainty: measurement and informational uncertainty. Measurement uncertainty refers to the variance that can be attributed to sampling techniques and laboratory analysis of contaminants. For example,

this type of uncertainty is associated with analytical data collected for each site. The risk assessment reflects the accumulated variances of the individual values used.

Informational uncertainty refers to estimates of toxicity and exposure. Often, this gap is significant, such as the absence of information on the effects of human exposure to low doses of a chemical, the biological mechanism of action of a chemical, or the behavior of a chemical in soil.

Once the risk assessment is complete, the results must be reviewed and evaluated to identify the type and magnitude of uncertainty involved. Reliance on results from a risk assessment without consideration of uncertainties, limitations, and assumptions inherent in the process can be misleading. For example, to account for uncertainties in the development of exposure assumptions, conservative estimates must be made to ensure that the particular assumptions made are protective of sensitive subpopulations or the maximum exposed individuals. If a number of conservative assumptions are combined in an exposure model, the resulting calculations can propagate the uncertainties associated with those assumptions, thereby producing a much larger uncertainty for the final results. This uncertainty is biased toward over predicting both carcinogenic and noncarcinogenic risks. Thus, both the results of the risk assessment and the uncertainties associated with those results must be considered when making risk management decisions.

This interpretation is especially relevant when the risks exceed the point-of-departure for defining "acceptable" risk. For example, when risks calculated using a high degree of uncertainty are below an "acceptable" risk level (1E-6), the interpretation of no significant risk is straightforward. However, when risks calculated using a high degree of uncertainty are above an "acceptable" risk level (1E-4), a conclusion can be difficult unless uncertainty is considered.

Recent EPA guidance on risk assessment (EPA, 1992c and 1994f) requires risk assessors to use exposure and toxicity assumptions from the "high end" and the "central tendency" of their distributions. These values correspond to the RME and CTE scenarios. The RME

is conceptually the "high end" exposure above the 90th percentile of the population distribution but not higher than the individual in the population with the highest exposure. The CTE reflects the central (average) estimates of exposure.

6.6.1 Uncertainty in Selection of Chemicals of Concern

There is a minor amount of uncertainty associated with the selection of COPCs on the final risk values in the quantitative risk assessment. Conservative screening values were used to select COPCs; thus, it is unlikely that any contaminants that may pose a risk were eliminated from the risk assessment.

6.6.2 Uncertainty in the Exposure Assessment

Uncertainty in the exposure assessment arises for the methods used to calculate exposure point concentrations, determination of land use conditions, selection of receptors, and selection of exposure parameters. Each of these is discussed below.

Calculation of Exposure Point Concentrations. For media at some study areas, fewer than ten samples were available. This makes the estimation of the 95 percent upper confidence limit on the mean highly uncertain and, therefore, the maximum detected chemical concentrations were used to assess RME risks. As a result, the estimations of risk where maxima were used as exposure concentrations are most likely to be overstated because it is unlikely that potential receptors would be exposed to the maximum concentration over the entire exposure period.

Exposure Routes and Receptor Identification. Exposure routes and receptor groups were based on discussions with the EPA and CTDEP and on site visits. This may either under- or over-estimate the risks, with the final result dependent on how well the receptors were defined.

Selection of Exposure Parameters. Each exposure factor selected for use in this risk assessment has some associated uncertainty. Generally, exposure factors are based on surveys of physiological and lifestyle profiles across the United States. The attributes and activities studied in these surveys generally have a broad distribution. To avoid underestimation of exposure, EPA guidelines on the RME receptor were used that generally consist of the 95th percentile for most parameters. Therefore, the selected values for the RME receptor represent the upper bound of the observed or expected habits of the majority of the population.

Many of the exposure parameters were determined from statistical analyses on human population characteristics. Often the database used to summarize a particular exposure parameter (body weight) is quite large. Consequently, the values chosen for such variables in the RME scenario have low uncertainty. For many parameters for which limited information exists (dermal absorption of organic chemicals from soil), there is greater uncertainty.

Many of the quantities used to calculate exposures and risks in this report are selected from a distribution of possible values. For the RME scenario, the value representing the 95th percentile is generally selected for each parameter to ensure that the assessment bounds the actual risks from a postulated exposure. In order to estimate a central tendency estimate of exposure, EPA has suggested the use of the CTE receptor, whose intake variables are set at approximately the 50th percentile of the distribution. The risks for this receptor seek to incorporate the range of uncertainty associated with various intake assumptions. Many of the parameters were estimated using professional judgment, although EPA Region I provides some default parameters (EPA, 1994f).

6.6.3 Uncertainty in the Toxicological Evaluation

A toxicity evaluation is the hazard identification and dose-response assessment of a chemical. The hazard identification deals with characterizing the nature and strength of the evidence of causation, or the likelihood that a chemical that induces adverse effects in

animals will also induce adverse effects in humans. Hazard identification of carcinogenicity is an evaluation of the weight-of-evidence that a chemical causes cancer. Positive animal cancer test data suggest that humans contain tissue(s) that may also manifest a carcinogenic response; however, the animal data cannot necessarily be used to predict the target tissue in humans. In the hazard assessment of noncancer effects, however, positive animal data suggest the nature of the effects (the target tissues and type of effects) anticipated in humans.

Uncertainty in hazard assessment arises from the nature and quality of the animal and human data. Uncertainty is reduced when similar effects are observed across species, strain, sex, and exposure route; when the magnitude of the response is clearly dose-related; when pharmacokinetic data indicate a similar fate in humans and animals; when postulated mechanisms of toxicity are similar for humans and animals; and when the chemical of concern is structurally similar to other chemicals for which the toxicity is more completely characterized.

Uncertainty in the dose-response evaluation includes the determination of a slope factor for the carcinogenic assessment and derivation of an RfD or Reference Concentration (RfC) for the noncarcinogenic assessment. The slope factor is an upper bound estimate of the human cancer risk per milligram of contaminant per milligram of body weight per day. The RfD and RfC are estimates, with uncertainty spanning perhaps an order of magnitude, of daily exposure to humans that below which is likely to be without appreciable risk of adverse effect over a lifetime. Uncertainty is introduced from interspecies (animal to human) extrapolation, which, in the absence of quantitative pharmacokinetic or mechanistic data, is usually based on consideration of interspecies differences in basal metabolic rate. Uncertainty also results from intraspecies variation. Most toxicity experiments are performed with animals that are very similar in age and genotype so that intragroup biological variation is minimal, but the human population of concern may reflect a great deal of heterogeneity, including unusual sensitivity or tolerance to the COPC. Even toxicity data from human occupational exposure reflect a bias because only those individuals sufficiently healthy to attend work regularly (the "healthy worker effect") and

those not unusually sensitive to the chemical are likely to be occupationally exposed. Finally, uncertainty arises from the quality of the key study from which the quantitative estimate is derived and from the database.

For cancer effects, the uncertainty associated with dose-response factors is mitigated by assuming the 95 percent upper bound for the slope factor. Another source of uncertainty in carcinogenic assessment is the method by which data from high doses in animal studies are extrapolated to the dose range expected for environmentally exposed humans. The linearized multistage model, which is used in nearly all quantitative estimations of human risk from animal data, is based on a nonthreshold assumption of carcinogenesis. There is evidence to suggest, however, that epigenetic carcinogens, as well as many genotoxic carcinogens, have a threshold below which they are noncarcinogenic (William and Weisburger, 1991); therefore, the use of the linearized multistage model is conservative for chemicals that exhibit a threshold for carcinogenicity.

For noncancer effects, additional uncertainty factors may be applied in the derivation of the RfD or RfC to mitigate poor quality of the key study or gaps in the database. Additional uncertainty for noncancer effects arises from the use of an effect level in the estimation of an RfD or RfC, because this estimation is predicated on the assumption of a threshold below which adverse effects are not expected. Therefore, an uncertainty factor is usually applied to estimate a no-effect level. Additional uncertainty arises in estimation of an RfD or RfC for chronic exposure from less-than-chronic data. Unless empirical data indicate that effects do not worsen with increasing duration of exposure, an additional uncertainty factor is applied to the no-effect level in the less-than-chronic study. Uncertainty in the derivation of RfDs is mitigated by the use of uncertainty and modifying factors that normally range between 3 and 10. The resulting combination of uncertainty and modifying factors may reach 1,000 or more.

Class C carcinogens are classified as possible human carcinogens because the evidence for their carcinogenicity in animals is limited. The inclusion of these compounds in the

estimation of total carcinogenic risk adds to the uncertainty of the final risk numbers by potentially overestimating the human health effects.

The derivation of dermal RfDs and CSFs from oral values may cause uncertainty. This is particularly the case when no gastrointestinal absorption rates are available in the literature or when only qualitative statements regarding absorption are available.

Uncertainty also arises in the dose-response assessment for values derived for several principal chemicals of concern by using studies with limitations. For example, Class B2 PAHs for which no toxicity data are available are evaluated using benzo(a)pyrene toxicity data with estimated orders of potential potency for the average and RME receptors. This may either underestimate or overestimate the carcinogenic risks associated with PAHs.

Uncertainty is associated with the exclusion of copper from the quantitative risk assessment. EPA Region I does not generally quantitatively evaluate non-carcinogenic hazards posed by copper because the toxicity value (RfD) has not been verified by EPA. Copper is a major contaminant at these sites. Exclusion of copper from this risk assessment may result in an under estimate of non-carcinogenic risks.

The carcinogenicity of arsenic via ingestion is not confirmed by the available data. However, EPA has proposed an oral unit risk factor that was used for all oral and dermal exposures to arsenic at this site. Since arsenic is a major risk driver, the risks may be overstated.

Some uncertainty is associated with the evaluation of chromium, which was assumed to be present in its hexavalent state. Since hexavalent chromium is considered to be more toxic than the trivalent state, which is essentially more common, risks for this chemical are probably overestimated to some degree.

Uncertainty in final calculations of risk results from assumptions made regarding additivity of effects from exposure to multiple compounds from various exposure routes. High

uncertainty exists when cancer risks for several substances are summed across different exposure pathways. This assumes that each substance has a similar effect and/or mode of action. Often compounds affect different organs, have different mechanisms of action, and differ in their fate in the body, so additivity may not be an appropriate assumption. However, the assumption of additivity is made to provide a conservative estimate of risk.

Finally, the risk characterization does not consider antagonistic or synergistic effects. Little or no information is available to determine the potential for antagonism or synergism for the COPCs. Therefore, this uncertainty cannot be discussed for its impact on the risk assessment, since it may either underestimate or overestimate potential human health risk.

Uncertainty is associated with the evaluation of exposures to lead. Two methods have been used in this risk characterization to evaluate lead exposures. Exposures of children to lead are evaluated using EPA's IEUBK model. Uncertainty is associated with the use of default values for exposures to lead via pathways other than soil ingestion. The IEUBK model was developed based on children exposed in a residential scenario. Application of this model to the recreational scenarios at this site results in uncertainties beyond those already inherent in the model. Exposures of commercial workers to lead are evaluated by use of the EPA Technical Review Workgroup Model for lead. This approach focuses on estimating fetal blood lead concentrations in women exposed to lead contaminated soils in non-residential scenarios. Uncertainty is associated with estimation of maternal blood lead concentrations and with the relationship between maternal blood lead concentrations and fetal blood lead concentrations.

This risk characterization does not include a quantitative risk evaluation of the risks associated with potential receptor exposures to asbestos because of the lack of appropriate toxicity criteria. Uncertainty in the risk characterization is associated with the presence of asbestos-containing material, defined as material containing more than one percent asbestos (EPA Regulation 40 CFR Subpart M, Part 61). Asbestos is considered a potential inhalation hazard if it is "friable" (can be crumbled, pulverized, or reduced to powder) and, consequently, subject to entrainment/migration into the air. It is

chosen as a COPC based on the health effects exhibited in occupationally exposed workers in epidemiological studies. Inhalation studies in animals and humans indicate that exposure to asbestos fibers may lead to asbestosis, lung cancer, tumors of the thin membranes surrounding internal organs such as the pleural and peritoneal membranes (mesotheliomas), and an increased risk of extrathoracic cancers. Asbestos exposure leading to cancer depends not only on dose but on underlying risk factors such as smoking.

6.7 Baseline Human Health Risk Assessment - Area A-1- Morgan Francis Property

This section contains the baseline risk assessment performed for soil, wetland soil, sediment, and surface water exposures at Area A-1, the Morgan Francis Property. Section 6.7.1 provides an overview of Area A-1, the Morgan Francis Property, Section 6.7.2 contains a discussion of the selection of COPCs, Section 6.7.3 contains information on the potential receptors considered and the routes by which they might be exposed, Section 6.7.4 contains the numerical results of the risk assessment, and Section 6.7.5 presents site-specific uncertainties associated with the risk assessment.

6.7.1 Overview of Area A-1, the Morgan Francis Property

Area A-I, the Morgan Francis Property, encompasses a portion of Ferry Creek, some commercial properties, and State of Connecticut properties, and a triangle-shaped parcel of land between Ferry Boulevard and East Broadway Street. The site covers approximately 11.1 acres, including wetlands and the upper portion of Ferry Creek. This portion of Ferry Creek bisects Area A-1, which is primarily used for commercial purposes. Further details of the site land use are presented in Section 6.7.3.1. The nature and extent of the contamination detected in Area A-1 was discussed in Section 4. Descriptive statistics (frequency of detection, range of positive detections, range of non-detects, location of maximum detections, and arithmetic mean) for target analytes detected in the Area A-1 environmental media are summarized in Tables 6-12 through 6-15.